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PLUTONIUM, ITS BIOLOGY AND ENVIRONMENTAL PERSISTENCE

by

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PLUTONIUM, ITS BIOLOGY AND ENVIRONMENTAL PERSISTENCE

INTRODUCTION

Perhaps no single element has ever been so intensively studied as has plutonium (Pu) in the 19 years since the first microgram quantities of this, the first of the man-made elements, became available in the summer of 1942. Literally thousands of reports have been published on the physical and chemical properties of the element and its isotope. Equally impressive is the number of reports concerning metabolism and toxicology of Pu in animals.

Several reasons may be cited for this tremendous effort. Pu, specifically the mass 239 isotope is superior as a nuclear fuel. For this reason, it is destined to play an increasingly important role in human affairs. Its chemistry is a complex and fascinating area of study. It is also one of the most toxic substances known to man, and consequently is of interest and concern to biologists, and to the medical profession.

Chemistry and Radioactive Properties

Plutonium is a member of the actinide series of rare earth elements. In general its chemistry is qualitatively similar to that of the rare earth. It forms an insoluble fluoride and hydroxide and a quite insoluble oxide. It can exist in several valence states, most commonly as the tri- and tetravalent cations or in the hexavalent form PuO_2^{+2}. It forms soluble complexes with citrate, and is strongly chelated. This last property has been used extensively in radiochemical separation by the now classical TTA extraction procedure. All possible isotopes between Pu232 and Pu246 are known and have been characterized. We will be

concerned almost exclusively with Pu239.

Pu239 decays by the emission of 5.15 MeV Alpha particles. These have a range of about 40 μ in water or soft tissue and about 24 μ in bone. It also emits x-rays with energies between 10 - 22 keV, and a very few 380 keV gamma rays. It has a half life of 24,400 years and a specific activity of 0.0617 curies per gram.

Metabolism and Distribution of Plutonium in Animal Tissues

While it is beyond the scope of this presentation to review extensively the literature pertaining to the biology of Pu, we would like to briefly summarize the present status of knowledge. Recent reviews by Langham (1959), Thompson (1960), and Williams (1959) present more comprehensive evaluations.

Oral Administration

Ingestion is generally considered as a minor source of metabolized Pu in mammals. Scott et. al. (1948) reported no significant absorption of ingested Pu. Katz, Kornberg, and Parker (1955) found after 517 chronic low level feedings of Pu238 to rats that less than 0.003% of administered dose was retained. In later studies, Weeks et. al. (1956) found this to hold true when Pu^{+4} nitrate in pH2. solution was fed. If more acid solutions were fed, or if present as PuO_2^{+2} a higher retention was found. Carritt et. al. (1947) found about 0.01% retention of orally administered Pu by rats after 5 daily feedings. Up to 0.3% was retained when administered as the citrate complex. Ballou and Kawin (1956) found that young rats absorb more Pu than do older animals.

Inhalation

Inhalation is considered as the primary route of entry for Pu into mammals. There is, however, lack of agreement as to the extent to which Pu entering the lung as inhaled particles is absorbed and deposited in other body tissues. Langham (1939) suggests that 10% may be absorbed into the general circulation, and that an additional amount may be phagocytized, thus entering the lymphatic circulation with ultimate deposition in lymph nodes. Bair (1958) presents data which indicate that absorption is about 1% from inhaled PuO_2 particles. The 10% figure may perhaps be considered a conservative upper limit.

Other Modes of Entry

Particularly in the case of persons working in processing plants or in laboratories handling Pu, there is a probability of contamination of intact skin or its introduction into wounds. The evidence from experiments conducted to evaluate these potential modes of entry generally indicate low degree of absorption. Langham (1959) reports that about 0.0002% of Pu239 in 0.4 N Nitric acid applied to the palm of a human subject was absorbed per hour of exposure. Wilson (1958) and Hammond (1958) present data from accident cases in which particulate Pu was introduced into superficial skin injuries. Most of the material remained localized at the site of the injury serving as a long term source for slow absorption. Scott et. al. (1948) showed a slow rate of translocation from the site of intramuscular injection to other tissues.

Deposition

Once Pu enters the blood stream it is rapidly and almost quantitatively deposited in various body tissues. Regardless of chemical state or of mode

of entry, the two major sites of deposition are the skeleton and the liver. Very significant quantitative differences do occur as functions of these variables. Katz et. al. (1955) found about 90% of Pu absorbed from the digestive tract to be deposited in bone, with only 10% in the balance of the animal. Following intravenous injection of Pu^{+4} complexed with citrate, Stover, Atherton and Keller (1959) found about 65% of the administered dose in bone, 22% in liver and 2 - 3% in blood and other tissues several weeks after injection. Caritt et. al. found essentially the same distribution following injection of Pu as the citrate complex or as PuO_2^{+2}. When uncomplexed tri- or tetravalent plutonium was injected, skeletal deposition was reduced while liver deposition was enhanced. This was considered to be due to formation of colloids in the blood stream. This interpretation is consistent with the observations of Schubert et. al. (1960) who found that only monomeric Pu is deposited in the skeleton, while colloidal polymeric forms are deposited in the liver. Following inhalation exposure, the absorbed Pu is deposited in other tissues with a similar pattern between bone and liver as for other modes of entry. The lungs retain a large part of the inhaled dose for considerable periods of time. Pulmonary lymph nodes characteristically show relatively high concentrations (Foreman, Moss and Langham, 1960; Bair, Willard and Hennacy, 1960; Bair, Willard and McClanahan, 1960).

In terms of micro-deposition and relative tissue concentrations, the foregoing generalized pattern is somewhat misleading. Bone deposited Pu is highly localized on the surface of recently deposited mineral. Bair et. al. (1959) found lymph nodes, spleen and ovaries to contain the highest

concentrations of Pu exclusive of lung following inhalation exposure.

Excretion and Turnover

The elimination of Pu from a mammalian system once it has gained entry is at best a slow process. Following inhalation, the elimination of non-absorbed particles is described by a power function of the general form $y = aT^{-b}$ in which y is the fraction excreted, a and b are constant and T is time in days (Bair, 1958). Bair, Temple and Willard (1959) and Willard (1960) present data indicative of the overall rate of elimination of inhaled PuO_2 particles by dogs. Two weeks after exposure, well over 50% of the dose remained in the lungs. Based on excretion rates over the period from 10 - 40 weeks after inhalation, Bair et. al. (1960) estimated that the half time for total body retention of inhaled Pu in dogs was about 1800 days. Even longer effective half times may be considered applicable to Pu deposited in various tissues. The effective half time in man has been estimated by Langham (1959) to be about 200 years. One might infer from this that under conditions of chronic exposure, that no equilibrium would be attained during a lifetime, and that tissue burdens would continue to increase indefinitely.

A number of studies have been conducted in search of chemical agents that would be effective in minimizing deposition or increasing excretion rates of previously deposited Pu. Langham (1959) cites evidence for the efficacy of zirconium citrate. Various chelating agents have been tried with varying degrees of success. Hamilton and Scott (1953) found ethylenediaminetetraacetic acid (EDTA) to be ineffective in removing Pu from bone. Diethylenetriaminepentaacetate (DTPA) was found by Ballou and Smith

(1960) to reduce skeletal retention by a factor of 10 when administered promptly following intravenous injection of Pu. DTPA was found superior to EDTA in removing Pu from bone by Lindenbaum, Westfall and Schubert (1960) and to reduce significantly the incidence of tumors as well as fairly effectively removing Pu from bone and liver by Rosenthal et. al. (1960).

Plutonium Toxicity

In terms of chemical toxicity, Pu would be expected to be comparable to uranium because of the great similarities in chemical properties. Because of its relatively high specific activity compared to uranium, even the 24,000 year Pu239 must be considered as primarily a radiotoxic agent. Pu levels which would produce symptoms of chemical toxicity would undoubtedly result in very significant radiation damage.

As a radiotoxin, Pu is generally considered to be extremely effective because the alpha particles have a limited range in tissue and produce an extremely high specific ionization. The characteristically non-homogeneous distribution in tissue further accentuates this. In reviewing her own work on long term effects of internally deposited isotopes, Finkel (1959) shows Pu239 to be more effective than Ra226 and U233, both of which also decay by Alpha emission, in shortening life span and in induction of bone tumors. Similarly, Stover et. al. (1959) find Pu239 more effective in tumor formation than Ra226. Alpha emitting isotopes in general are considered to be about 20 times as effective as beta or gamma emitters in terms of biological reactions.

We will not attempt here to review the extensive literature pertaining to Pu toxicity. The Hanford Biology Group has for many years been engaged

in toxicity studies. Warner (1960) presents a listing of reports from that laboratory. For the past 10 years a major project in chronic toxicity studies has been in operation at the University of Utah. Stover (1958, 1959a, 1959b) and Dougherty (1960a, 1960b) present details of their studies. Significant contributions have been made by other laboratories.

Among the manifestations of Pu toxicity that have been reported are shortening of life expectancy (Finkel, 1959), bone tumors (Finkel, 1959; Stover, 1958, 1959a, 1959b; Dougherty, 1960a, 1960b), lung tumors (Lisco, 1959; Temple, Marks and Bair, 1960), reduced resistance to gamma radiation (Ballou, 1959, 1960), lymphatic pathologies (Temple and Bair, 1960), reduced lymphocyte counts and serum albumin levels (Clarke, McKenney and Horstman, 1960), ovarian damage (Vogt and Kawin, 1958; Bloom, 1948) and superficial epithelial damage in intestine (Sullivan et. al., 1960).

Plutonium in the Environment

Plutonium has been a part of our environment throughout history in extremely minute amounts. Seaborg (1958) cites considerable evidence for the presence of a few parts of Pu in 10^{12} parts of uranium (micrograms/ton of uranium). Measurable amounts have been present in soils and biological materials only since the first man-made Pu was released to the environment on July 16, 1954. It has been estimated by the United Nations Scientific Committee on the Effects of Atomic Radiation (1959) that all nuclear tests conducted to the end of 1958 have resulted in the formation of 5×10^5 curies of 8×10^6 grams of Pu239. By comparison,

they estimated that Sr90 and Cs137 have been formed to the extent of 92 $\times 10^5$ and 135 $\times 10^5$ curies respectively.

Data reflecting cumulative deposition of Pu239 are virtually non-existent. Stewart, Crooks and Fisher (1956) estimated that as of the end of 1955, levels in the United Kingdom were about 0.2 of the then existing Sr90 levels. Recent data obtained in this country on the current rate of deposition of fallout radioactivity show that Pu239 levels presently appearing in fallout collectors are at most a few per cent of the coincident Sr90 levels (Hardy, Rivera and Frankel, 1961a, 1961b, 1961c). The evidence at hand suggests that the general environmental contamination with Pu239 is at least one order of magnitude less than the Sr90 levels of which most of us are aware. There are relatively limited areas at or near nuclear test sites where much higher levels occur. It is from studies at these sites that much of the limited ecological data has been obtained.

Once deposited on the soil, fallout Pu239, presumably as the insoluble oxide, and incorporated with other bomb and soil debris, appears to be inert and to remain very near the soil surface of long periods of time. Olafson, Nishita and Larson (1957) show that for the first 11 years following the Trinity test in New Mexico, surface soil levels remained unchanged with little or no evidence of downward migration in an area with good vegetative cover to minimize erosion.

Atmospheric Pu239

Since inhalation is recognized as the main route of entry into mammals, the levels of airborne Pu239 are of prime concern. The only

data we have found relating to concentration in air away from highly contaminated areas at the Nevada Test Site and the Trinity Site in New Mexico were obtained in Japan in early 1957. Hiyama (1957) found from 1 - 2.5 x 10^{-8} pC of Pu239 per liter of air during the first half of that year. (1 pico curie, pC, is $10.^{-12}$ curies of 2.22 dis/min). This is equivalent to a maximum of 2.5 x 10^{-11} μc/cubic meter and is an insignificant level compared to the MPC value of 4 x 10^{-5} μc/cubic meter for occupational exposure to insoluble Pu (N.B.S., 1959).

In April, 1957, a non-critical high explosive detonation involving Pu was set off at the Nevada Test Site to evaluate various problems that might arise following an accidental release. During the first 3 hours following that event, mean airborne levels up to 35000 dis/min/M^3 were observed. (Shreve, 1958). These early levels were initially measured by gross alpha counting. The observed concentrations decreased rapidly. By 7 hours the observed values had decreased by a factor of 100, and by a factor of 500 at 20 days. It was estimated on the basis of these data that only a very small area at the immediate site would have produced above tolerance levels in man. Beginning 18 days after this event and continuing to 160 days, Wilson Thomas and Stannard (1960) followed the airborne concentrations in this area. A summary of their data is presented in Table 1. They have extrapolated their data back to time of placement of experimental animals 2 days post shot. The total activity figures represent observed or extrapolated values measured on their filters. The values for inspired activity represent their estimates of what actually was deposited in the lungs of experimental dogs exposed

Table I. Summary of Exposure Period Air Sampler Information (data from Wilson et. al., 1960)

Days after Placement	Total air Sampled	Pu-239, disintegrations/minutes					
		10 µg/M^2		100 µg/M^2		1000 µg/M^2	
	liters	total	inspired	total	inspired	total	inspired
4	9.6×10^4	313	31	857	86	2277	228
8	1.9×10^5	620	62	1653	165	4507	451
16	3.9×10^5	1149	115	2846	285	8356	836
32	7.8×10^5	2000	200	4761	476	14543	1454
64	1.6×10^6	3088	309	7197	720	22449	2245
96	2.4×10^6	3667	367	8553	855	26050	2605
128	3.1×10^6	3984	398	8995	900	28964	2896
160	3.9×10^6	4151	415	9350	935	30178	3018

at the air sampling locations. They assumed a breathing rate of one fifth of the air sampling rate and that half of the particles sampled were of respirable size. We have recalculated their data to indicate the mean airborne concentrations over the various time increments. Table 2 shows these values expressed as the ratio observed concentration/MPC for occupational exposure.

During the summer of 1958 our laboratory conducted studies of airborne concentrations of Pu239 at the same site (Larson, 1958). Sampling was carried on continuously for 21 days with filters being replaced every 12 hours. Each filter was analyzed by our standard procedure (Olafson et. al., 1957). Typical data are presented in Table 3 along with soil deposited levels at the sampling locations. The two high level areas are

Table II. Time: Concentration Relationship of Airborne Pu-239 Following Release to the Environment (data from Wilson et. al., 1960)

Time increment after placement days	Volume sampled, M^3	Ratio Observed/MPC insoluble Pu $10\ \mu g/M^2$	$100\ \mu g/M^2$	$1000\ \mu g/M^2$
0 - 4	96	.037	.10	.265
4 - 8	96	.037	.095	.265
8 - 16	190	.032	.070	.225
16 - 32	390	.025	.055	.180
32 - 64	800	.016	.034	.110
64 - 96	800	.008	.013	.050
96 - 128	800	.0045	.006	.040
128 - 160	800	.0025	.005	.017

essentially the same locations as the nominal 100 and 1000 μgm. locations of Wilson et. al. The low level site was about four miles from the detonation site. The maximum observed concentration was about 12% of the occupational MPC with most values at 1% or less. Considerable daily variation as a function of weather conditions is apparent, as well as a general lack of agreement between the levels of deposited Pu and airborne concentrations.

Pu239 in plants and other foods

Although ingestion is a minor source of Pu in mammalian tissues, it is nevertheless desirable to consider the passage of Pu from soil through the food chain leading to man.

Pu is absorbed by plant roots and translocated to aerial portions of the plant to only a very slight degree. Overstreet and Jacobson (1948) found that 0.01% of PuO_2^{+2} was absorbed and translocated from a culture solution to which the Pu was added as a bentonite suspension. Less than

Table III. Airborne Concentrations of Pu-239
at Release Site in Nevada, 1958

	Soil Activity, $\mu g/M^2$		
	782	67.6	.74
Date	Ratio observed air conc./MPC occupational for Insol. Pu		
7/26	$.8 \times 10^{-2}$	2.5×10^{-4}	0
7/27	1.0	5.0	0
7/28	12.2	19.0	$.46 \times 10^{-5}$
7/29	5.3	20.7	4.5
7/30	6.8	3.2	1.8
7/31	1.8	0.4	23.
8/1	1.4	9.3	0

20% was adsorbed on the clay. Pu^{+3} which was initially adsorbed to the clay to the extent of over 90% was translocated only to the extent of 0.0045%. Exposure time was 24 hours. Using a Neubauer seedling test, Rediske, Cline and Selders (1955) found the concentration in the plants to be 9×10^{-4} that of the soil. Romney (1961) at our laboratory is conducting a prolonged cropping study of Pu239 uptake by ladino clover from a Nevada Test Site soil heavily contaminated by non-critical high explosive shots. Data just obtained indicate a plant to soil concentration ratio of about 1×10^{-4}. Twelve crops harvested between March, 1958 and April, 1961 have removed from the soil only about 2×10^{-5}% of the total present. This evidence all indicates a very low degree of uptake, although the chemical state appears to be a governing factor.

The Atomic Energy Commission (1959b) presented a summary of data then available showing concentrations of Pu in various food items and plant materials. These are summarized in Table 4. The concentrations in

Table IV. Plutonium Concentrations in Various Food Items and Plants (data from USAEC, 1959)

	Pu239 pC/g.
Rain	.000178 ± .000027
Alfalfa Ash	.432 ± .042 to .800 ± .077
Milk	.00016 ± .00013
Wheat Ash	.127 ± .023 to .672 ± .045
Swordfish	.00034 ± .00025 to .0010 ± .00032
Pork Liver	.00056 ± .00026 to .00273 ± .00029
Beef Meat	.00019 ± .00013 (meat of chuck steak)
	.184 ± .0095 (fluid)

all items except the plant materials are extremely low and would contribute negligible amounts of Pu to human tissue burdens. The relatively high levels in the alfalfa and wheat ash are most probably due to external contamination.

Pu239 in Mammalian Tissues

In line with the preceding discussion, concentrations of Pu239 in mammalian tissues due to environmental contamination are generally quite low. The information that is available does, in general, permit some evaluation in terms of the extent to which the environmental Pu is entering the mammalian systems. This is possible since most of the studies have involved determinations of concentrations in soils and air at the collection site.

The earliest evidence for the presence of Pu239 in animal tissues was presented by Larson et. al. (1951) and by Leitch (1951). Based on gross alpha counting of ashed tissues of rodents trapped in the fall-out zone of the Trinity shot in 1949 and 1950, an alpha activity assumed

Table V. Median Pu-239 Concentrations in Dog Tissues
(data from Wilson et. al., 1960)

Exposure condition	Dis/Min/g. West Tissue GI tract	Hilar lymph nodes	Mediastinal lymph nodes	lung	femur	rib
Acute	25	2.05	3.5	.75	.046	.56
1000 $\mu g/M^2$	4.5	2.05	4.9	.45	.057	.70
100 $\mu g/M^2$	1.1	3.32	6.9	.020	.057	.46
10 $\mu g/M^2$	.35	3.05	3.5	.031	1.13	.19

to be Pu239 was observed in animals from one area, 25 - 28 miles from the detonation site. Unpublished data obtained in 1956 at our laboratory verifies the presence of Pu239 in bone of animals in that area to the extent of about .05 disintegrations per minute per gram of ash. The soils in that area contained about 10 μg per square meter, virtually all in the surface inch (Olafson et. al., 1957). Airborne concentrations were measured in the area in 1950 and were found to be less than 7.5 x 10^{-9} μc per cubic meter (Larson et. al., 1951).

The accumulation of Pu239 by experimental animals over a period of 160 days following release was investigated by Wilson et. al. (1960). A representative portion of their data is presented in Table 5. The relatively high concentration in lymph nodes and lungs is indicative that inhalation was the main route of entry. Undoubtedly the animals ingested some Pu with their diet, but relatively little of the tissue burden can be attributed to this. The surprising aspect of their data is that the acute exposure animals which were killed within a few hours to 4 days, had essentially the same concentrations as the chronically

Table VI. Estimated Effective Dose Rates to Man Based on Median Dog Values (data from Wilson et. al., 1960)

Exposure condition	Effective dose, rem/week: GI tract	Hilar lymph nodes	Mediastinal lymph nodes	lung	rib
Acute	.0055	.00045	.0008	.0031	.013
1000 $\mu g/M^2$	.001	.00045	.0016	.0020	.018
100 $\mu g/M^2$	.00025	.00075	.0014	0	.011
10 $\mu g/M^2$	.00006	.0007	.0008	.00014	.0043

exposed animals. This appears to be contrary to expectations if one assumes that the Pu was dispersed as oxide particles. From these dog data they estimated an effective dose to several tissues in man under comparable exposure conditions. These are shown in Table 6. In arriving at these values they assumed energy degradation by a factor of 20 for the intestinal tract and lymphatic tissue. Because non-uniform distribution in lung and bone results in high local dose levels, no energy degradation correction was made, and the tabulated values reflect the maximal dose estimates. They, however, go on to point out that, if only analytical values greater than 2 disintegrations per minute, which is the apparent sensitivity of the procedure used, are considered as significant then only the digestive tract and lung could be considered as receiving a significant dose.

During the summer of 1958 our laboratory collected for Pu assay small rodents and jack rabbits in this area (Larson, 1958). A summary of our analytical data is presented in Tables 7 and 8. All tissues

Table VII. Mean Pu-239 Concentration in Jack Rabbit Tissue One Year After Pu Release Test

Soil Pu $\mu g/M^2$	No. of animals	GI tract	Lung	Bone
		d/min/tissue		d/m/gm. Ash
782	6	154,000	57.5	2.2
67.6	7	378	.36	.02
2.5	5	672	.03	.03
.74	9	671	.45	.003
.74	38	293	.11	.01
.36	10	59	.14	.03

Table VIII. Mean Pu-239 Concentrations in Kangaroo Tissues One Year After Pu Release from Safety Test

Soil $\mu g/M^2$	No. of animals	GI tract	Lung	Liver	Bone
		d/min/tissue			d/m/gm.ash
67.6	20	1744	.01	.15*	1.58
2.5	5	349	.01	.16**	.07
.74	6	186	.01		.05
.74	16	151	.07		.05
.36	10	50	0		.03

* - 3 animals

** - 2 animals

analyzed were found to contain traces of Pu with digestive tracts containing very significant concentrations in all cases. If one assumes that the activity found in digestive tracts represents a mean daily ingestion over a period of months, the small amount of tissue deposited Pu could be attributed to ingestion. At the same time, inhalation cannot be ruled out as the source since low but readily detectable levels were found in many of the lungs. Both routes of entry probably contribute. While it appears that these animals, living very close to the soil, get at least a very significant part of their tissue burden by way of ingestion, it does not alter the general conclusion that inhalation is the main route of entry in humans.

Measurable but very low concentrations of Pu239 have been found in human tissues. Data obtained prior to 1959 were presented by the AEC (1959a) as part of a statement regarding fallout to the Congressional Hearings in June of that year. These are shown in Table 9. While all concentrations are very low, they represent measurable levels. The relatively high concentration in lymph nodes is indicative of inhalation of the mode of entry. The gonad concentrations have attracted attention and raised the question of which are truly the critical tissues for Pu deposition.

Table IX. Pu-239 Concentrations in Human Tissues (data from AEC, 1959a)

	pC/gram
Bone ash	Bkg
Lung	.00536 ± .00049
Tracheo - hair nodes	.0292 ± .014
Composite Organs	
Lung	.00784 ± .000077
Pulmonary nodes	.0049 ± .0018
Spleen	.000518 ± .000095
Kidney	.00044 ± .000090
Gonad	.0035 ± .00086

Summary and Conclusions

Even a cursory review of the literature pertaining to plutonium and the manifold manifestations of its toxicity leaves little doubt that it is an extremely hazardous material. Every effort should be made to minimize the extent to which man is exposed to it.

Plutonium as an environmental contaminant would not, on the basis of the evidence presently available, appear to be a serious problem. Since it decays by emission of alpha particles it presents no hazard in terms of external whole body radiation. It is absorbed by plants growing on contaminated soil to an infinitesimal degree, although it may be found as an external contaminant on vegetation. Ingested Pu is absorbed and retained in animal tissues to a very small degree. Therefore, relatively high levels in dietary constituents can be tolerated without producing a ponderable tissue burden. Extremely high levels of

Pu239 are required to produce even superficial tissue damage in the digestive tract. Inhalation of airborne particulates remain as the most effective means of entry into higher animals including man. But for this mechanism to produce a tissue burden, there must be significant airborne concentrations. The available information indicates that even in localized regions having very high levels of deposited Pu239 that the levels in air are, except for accasional periods, low in comparison to what is considered as tolerable concentrations for occupational exposure. Animal tissue assays have verified that very little Pu gains entry into mammalian systems from the contaminated environment.

While no one should be alarmed about concentrations of Pu239 in the environment of man, or more specifically in human beings themselves, we would urge caution in drawing conclusions regarding ecological implications. Are the present concentrations only the beginning of a continuously rising trend? This is a possibility because of extremely slow turnover rate and long half life. Will the apparent tendency for Pu concentrations in reproductive tissue continue on a chronic basis following acute exposure? If so, will such tissues in time have concentrations of genetic significance? These are only a few of the obvious questions to which answers are needed before a final verdict is reached. While the prognosis is encouraging, based on our present knowledge, a much greater understanding of the whole complex of environmental reactions is needed.

Literature Cited

Bair, W. J., 1958. Translocation and excretion of pulmonary deposited plutonium oxide. Document HW-56636.

Bair, W. J., L. A. Temple and D. H. Willard, 1959. Deposition and translocation of radioactive particles. Document HW-60137, pp 8-9.

Bair, W. J., D. H. Willard and R. A. Hennacy, 1960. Radioactive particles. Document HW-63643, pp 10-11.

Bair, W. J., D. H. Willard and B. J. McClanahan, 1960. Excretion and translocation of $Pu^{239}O_2$ after inhalation. Document HW-65500, pp 110-115.

Bair, W. J., D. H. Willard, L. A. Temple and G. D. Smith, 1959. Behavior of inhaled radioactive particles in mice. Document HW-59500, pp 101-105.

Ballou, J. E., 1959. Synergistic effects of internally deposited plutonium and external x-irradiation. Document HW-59500, pp 61-62.

Ballou, J. E., 1960. The effect of combined plutonium and x-irradiation in the rat. Document HW-65500, pp 147-153.

Ballou, J. E. and B. Kawin, 1956. Plutonium absorption and metabolism. Document HW-46333, pp 10-11.

Ballou, J. E. and V. H. Smith, 1960. Effectiveness of DTPA in removing Pu from swine. Document HW-65500, pp 78-81.

Bloom, W., 1948. Histopathology of irradiation from external and internal sources. In National Nuclear Energy Series, Division IV, Vol. 221, pp 596-611.

Carritt, J., R. Fryxell, J. Kleinschmidt, R. Kleinschmidt, W. Langham, A. San Pietro, R. Schaffer and B. Schnage, 1947. The distribution and excretion of plutonium administered intravenously to the rat. J. Biol. Chem. 171: 273-283.

Clarke, W. J., J. R. McKenney and V. G. Horstman, 1960. Comparative toxicity of Sr90, Ru226 and Pu239 in miniature swine. Document HW-65500, pp 53-55.

Dougherty, R. F., 1960a. Research in radiobiology. Annual report. Document 000 220.

Dougherty, T. F., 1960b. Research in radiobiology. Semiannual report. Document 000 222.

Finkel, M. P., 1959. Late effects of internally deposited radioisotopes in laboratory animals. Rad. Research, Supl. 1, pp 265-279.

Foreman, H., W. Moss and W. Langham, 1960. Plutonium accumulation from long term occupational exposure. Health Phys. 2: 326-333.

Hamilton, J. G. and K. G. Scott, 1953. The effects of CaEDTA upon the metabolic pattern of plutonium in rats. Proc. Soc. Exp. Biol. Med. 83: 301-305.

Hammond, S. E., 1958. Plutonium excretion following acute accidental exposure. In "Proceedings of the Fourth Annual Meeting in bio-assay and Analytical Chemistry", Nov. 3 and 4, 1958. Document WASH 1023, pp 84-100.

Hardy, E. P., Jr., J. Rivera and R. Frankel, 1961a, b, c. Fallout program, Quarterly Summary Report, Documents HASL 105, HASL 111, HASL 113.

Hiyama, Y., 1957. Radiological data in Japan. II. Concentrations of Sr-90, Cs-137 and Pu-239 and others in various materials on earth's surface. Document A/AC.82/G/R.141.

Jacobson, L. and R. Overstreet, 1948. The uptake by plants of plutonium and some products of nuclear fission adsorbed on soil colloids. Soil Science 65: 129-134.

Katz, J., H. A. Kornberg and H. M. Parker, 1955. Absorption of plutonium fed chronically to rats I. Fraction deposited in skeleton and soft tissue following oral administration of solutions of very low mass concentration. Am. J. Roentgenol. 73: 303-308.

Langham, W. H., 1959. Physiology and Toxicology of plutonium 239 and its industrial medical control. Health Physics 2: 172-185.

Larson, K. H. 1956. Unpublished data.

Larson, K. H., 1958. Unpublished data.

Larson, K. H., J. L. Leitch, W. F. Dunn, J. W. Neel, J. H. Olafson, E. E. Held, J. Taylor, W. J. Gross, A. W. Bellamy and L. Baurmash, 1951. Alpha activity due to the 1945 atomic bomb detonation at Trinity, Alamogordo, New Mexico. Document UCLA-111.

Leitch, J. L., 1951. Summary of the radiological findings in animals from the biological surveys of 1947, 1948, 1949 and 1950. Document UCLA-108.

Lindenbaum, A., W. M. Westfall and J. Schubert, 1960. Progress report: Plutonium Removal III. Effects of chelating agents on the ultrafiltration of intravenously administered plutonium from plasma. Document ANL-6200, pp 89-90.

Lisco, H., 1959. Autoradiographic and bistopathologic studies in radiation carcinogenesis of the lung. Lab. Invest. 8: 162-170.

National Bureau of Standards, 1959. Maximum permissible body burdens and maximum permissible concentrations of radionuclides in air and water for occupational exposure. Handbook 69.

Olafson, J. H., H. Nishita and K. H. Larson, 1957. The distribution of plutonium in the soils of central and northeastern New Mexico as a result of the atomic bomb test of July 16, 1945. Document UCLA-406.

Rediske, J. H., J. F. Cline and A. A. Selders, 1955. The absorption of fission products by plants. Document HW-36734.

Romney, E. M. 1961. Unpublished data.

Rosenthal, M. W., J. F. Fried, A. Lindenbaum and J. Schubert, 1960. Progress Report: Relationship of plutonium removal to tumor incidence. I. Experimental design and preliminary results. Document ANL-6264, pp 172-176.

Schubert, J., J. F. Fried, M. W. Rosenthal and A. Lindenbaum, 1960. Progress Report: Plutonium removal. IV. Tissue distribution of monomeric and polymeric Pu as modified by a chelating agent. Document ANL-6264, pp 177-178.

Scott, K. G., D. J. Axelrod, H. Fisher, J. F. Crowley, and J. G. Hamilton, 1948. Metabolism of Pu in rats. J. Biol. Chem. 176: 283-293.

Seaborg, G. T., 1958. The transuranium elements. Yale University Press, New Haven 328 p.

Shreve, J. E., Jr., 1958. Summary report, Test Group 57, Operation Plumbbob, Preliminary report, Document ITR-1515 (Del).

Stewart, N. C., R. N. Crooks and E. M. R. Fisher, 1956. The radiological dose to persons in the U. K. due to debris from nuclear test explosions prior to January 1956. Document AERE/HP/R-2017.

Stover, B. J., D. R. Atherton and N. Keller, 1959. Metabolism of Pu-239 in adult beagle dogs. Radiation research 10: 130-147.

Stover, C. N., Jr., (Ed.), 1958. Semi annual progress report. Document 000-217.

Stover, C. N., Jr., (Ed.), 1959a. Annual progress report. Document 000-218.

Stover, C. N., Jr., (Ed.) 1959b. Semi annual progress report, Document 000-219.

Sullivan, M. F., P. L. Hackett, L. A. George and R. C. Thompson, 1960. Irradiation of the intestines by radioisotopes. Radiation research. 13: 343-355.

Temple, L. A. and W. J. Bair, 1960. Biological effects of inhaled $Pu^{-239}O_2$ and $Ru^{-106}O_2$ in mice. Document HW-65500, pp 129-138.

Temple, L. A., S. Marks and W. J. Bair, 1960. Tumors in mice after pulmonary deposition of radioactive particles. Intern. J. Radiation Biol. 2: 143-156.

Thompson, R. C., 1960. Vertebrate radiobiology: Metabolism of internal emitters. Ann. Rev. of Nucl. Science 10: 531-560.

United Nations, Scientific Committee on the effects of atomic radiation, 1959. Status report of the food chain. Document A/AC.82/R.80.

United States Atomic Energy Commission, 1959a. Quarterly Statement on fallout. In fallout from Nuclear Weapons Tests. Congressional Hearings, pp 2188-2189.

United States Atomic Energy Commission, 1959b. Quarterly Statement on fallout. Document A/AC.82/C/L.277.

Vogt, G. and B. Kawin, 1958. Localization of radioelements in rat ovary. Document HW-53500,

Warner, D. E., (Ed.) 1960. Hanford Biology Research Annual Report for 1959. Document HW-65500, 206 p.

Weeks, M. H., J. Katz, W. D. Oakley, J. E. Ballou, L. A. George, L. K.

Bustad, R. C. Thompson and H. A. Kornberg, 1956. Further studies on the gastrointestinal absorption of plutonium. Rad. Research 4: 339-347.

Willard, D. H., 1960. Distribution and toxicity of inhaled $Pu^{-239}O_2$. Document HW-65500, pp 122-128.

Williams, K., 1959. Studies on the toxicology of plutonium. Document AERE-R-2970.

Wilson, R. H., 1958. A review of significant Hanford plutonium deposition cases. In "Proceedings of the Fourth Annual Meeting on Bioassay and analytical chemistry, No. 3 and 4, 1958". Document WASH-1023, pp 71-83.

Wilson, R. H., R. G. Thomas and J. N. Stannard, 1960. Biomedical and aerosal studies associated with field release of plutonium. Document WT-1511.